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Reply

Quantification of Atherosclerotic Coronary Plaque

The Missing Link Between Elevated Biochemical Markers and Adverse Outcomes in the "Vulnerable" Patient?

We greatly appreciate the comments made by Drs. Korosoglou and Katus regarding our paper (1). The authors acknowledge the promising potential of plaque quantification for individualized prediction of acute coronary syndrome (ACS). At the same time, highly-sensitive cardiac troponin T (hsTnT) also has important prognostic implications, although indeed the pathophysiologic mechanism is not fully understood. Previous results showed hsTnT levels to be associated with extent of coronary artery disease (2) and to have independent prognostic value on top of stenosis severity, as assessed by cardiac computed tomographic angiography (3). The suggestion that hsTnT elevation might be a marker of plaque instability is very interesting and could possibly explain these findings. We agree that the association between high-risk plaque features and hsTnT levels points toward this direction (4). Our current investigation is really a first step to identify the additional value of coronary plaque quantification, and it would be a very interesting next study to validate serum biomarkers again using high-risk imaging parameters.

The authors express some methodological concerns. Regarding selection bias, our objective was to investigate the potential additional value of plaque quantification over conventional cardiac computed tomographic angiography readings. In fact, we compared 2 methods within the same patient group. That is the reason why we chose a randomly-selected control population: the influence of selection bias would be irrelevant to this comparison. As mentioned by the authors, the fact that ACS can also occur in the absence of angiographically-visible coronary artery disease is really an intrinsic limitation of plaque assessment in general (and could limit its prognostic value in a prospective study). Unfortunately, predicting these elusive ACS cases remains challenging. The semiautomated nature of our quantification algorithm results in approximately 15 min operating time per patient and, indeed, some interobserver variability. For clinical practice, the development of a reliable automated method would be beneficial.

Most important, a prospective study on plaque quantification could validate our suggested high-risk parameters. This information could help us to identify a simple and clinically useful "risk score" based on imaging data. Combining coronary imaging and serum biomarkers (such as hsTnT) may possibly further improve individualized risk assessment. We fully agree with the authors that, regarding the wide array of novel therapeutic options, this would be a very important goal to pursue.

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